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EFFECTS OF TRANSCUTANEOUS ELECTRICAL NERVE STIMULATION (TENS) ON SPASTICITY IN PATIENTS WITH HEMIPLEGIA

Karmen Peterlin Potisk, MD,[1] Milan Gregorič, MD, PhD[1] and Lojze Vodovnik, Dipl Ing, PhD[2]

From the [1] Institute for Rehabilitation, and the [2] Faculty of Electrical and Computer Engineering, University of Ljubljana, Ljubljana, Slovenia

ABSTRACT. The effect of afferent cutaneous electrical stimulation on the spasticity of leg muscles was studied in 20 patients with chronic hemiplegia after stroke. Stimulation electrodes were placed over the sural nerve of the affected limb. The standard method of cutaneous stimulation, TENS with impulse frequency of 100 Hz, was applied. The tonus of the leg muscles was measured by means of an electrohydraulic measuring brace. The EMG stretch reflex activity of the tibialis anterior and triceps surae muscles was detected by surface electrodes and recorded simultaneously with the measured biomechanical parameters. In 18 out of 20 patients, a mild but statistically significant decrease in resistive torques at all frequencies of passive ankle movements was recorded following 20 min of TENS application. The decrease in resistive torque was often (but not always) accompanied by a decrease in reflex EMG activity. This effect of TENS persisted up to 45 min after the end of TENS. The results of the study support the hypothesis that TENS applied to the sural nerve may induce short-term post-stimulation inhibitory effects on the abnormally enhanced stretch reflex activity in spasticity of cerebral origin.

Key words: hemiplegia, spasticity, transcutaneous electrical nerve stimulation.

Electrical neuromuscular stimulation is used in the treatment and rehabilitation of the upper motor neuron syndromes to improve certain motor functions and restore functional movement. It was shown that electrical stimulation of nerves and muscles could exert a beneficial, suppressive effect on the abnormally increased tonic and phasic stretch reflex activity involved in spastic muscle hypertonia and clonus (1,4,14,16,17,20–22,25,26).

The thick myelinated cutaneous nerve fibres can be selectively stimulated by low intensity transcutaneous electrical nerve stimulation (TENS). Prolonged application of TENS induces an analgesic effect which is widely applied in clinical practice for the treatment of pain.

Earlier studies have demonstrated suppressive effects of TENS on the spastic muscle hypertonia and exaggerated flexor reflex responses in spinal spasticity (3, 8, 16). TENS applied repetitively to the common peroneal nerve has been found to reduce clinical spasticity and improve control of reflex and motor functions in hemiparetic subjects (12). TENS was shown to induce long-lasting changes in the motoneural excitability of the soleus muscle, parallel to the changes in sensory functions when stimulation was applied to the sural nerve (7). However, these findings were not confirmed by some other studies (9).

In the present study we wished to explore the hypothesis in which low intensity and high frequency transcutaneous electrical nerve stimulation, applied to the sural nerve, exerts post-stimulation suppressive effects on the exaggerated stretch reflex activity of the leg muscles in chronic spastic hemiplegia.

METHODS

Patients

Twenty hemiplegic patients, at least 3 months after stroke, were enrolled in the study. The patients' data are presented in Table I. Prior to the experiments, the patients had not received any treatment with electrical stimulation. Patients with diabetes or other types of polyneuropathy, those with ankle joint contractures and drug treatment for spasticity were not included in the study.

Electrical stimulation

A commercial battery-powered electrical stimulator, designed for analgesic electrotherapy was used in this study. Surface electrodes were applied over the sural nerve on the affected limb. The anode was at the level of the lateral malleolus and cathode 10 cm proximally. The stimulator delivers biphasic impulses with an amplitude of 0–100 V. The frequency of impulses was 100 Hz and the pulse width 0.2 msec. The intensity of stimulation was adjusted to a level just below the threshold for a visible muscle contraction.

Table I. *Clinical data on the patients*

Pat.	Age (years)	Sex	Stroke type	Diagnosis	Sensibility S	Sensibility P	Time after (months)
1	60	M	Ischemic	R hemiparesis	N	N	3
2	38	M	Ischemic	R hemiparesis	N	N	6
3	63	M	Ischemic	R hemiparesis	N	N	4
4	69	F	Ischemic	R hemiparesis	N	N	3
5	62	M	Ischemic	R hemiparesis	N	N	6
6	68	M	Ischemic	R hemiparesis	I	N	12
7	69	M	Haem	L hemiparesis	I	I	3
8	48	M	Haem	R hemiparesis	I	I	3
9	68	F	Ischemic	L hemiparesis	N	N	4
10	61	M	Ischemic	R hemiparesis	I	I	5
11	58	F	Haem	R hemiparesis	I	I	3
12	49	M	Ischemic	R hemiparesis	I	I	5
13	61	F	Ischemic	R hemiplegia	N	N	7
14	63	M	Ischemic	R hemiparesis	I	I	4
15	57	M	Ischemic	L hemiparesis	I	I	17
16	50	M	Ischemic	L hemiparesis	N	N	4
17	59	M	Ischemic	L hemiplegia	I	I	4
18	49	M	Haem	R hemiplegia	I	I	9
19	67	M	Ischemic	L hemiparesis	N	N	6
20	43	M	Ischemic	R hemiplegia	I	I	3

haem = haemorrhagic stroke
R = right; L = left
S = surface; P = proprioception; N = normal; I = impaired.

Resistive torque due to passive movement

Sinusoidal passive movements of the foot in the sagittal plane were applied for testing spastic muscle hypertonia. The joint movements were performed by an electrohydraulic position-controlled servosystem described by Reberšek (19). Spasticity of the leg muscles was assessed as a resistive torque due to passive sinusoidal movements. Velocity dependence of stretch reflex was tested at four different velocities of ankle joint movements, defined by frequencies of sinusoidal oscillations: 0.1, 0.5, 1 and 2 Hz. Ten successive periods of oscillation were applied at each frequency. Four parameters were recorded on Gould Brush 440: electromyographic activity (EMG) of the tibialis anterior and triceps surae muscles, position of the ankle joint, and resistive torque.

Electromyographic activity was detected by pairs of Ag/AgCl disc electrodes of 1 cm diameter, the distance between the electrodes being 3 cm. The electrodes were placed over the middle part of the muscle belly of the tibialis anterior and over the soleus muscle. The activity recorded over the latter was considered to represent partly also the activity of the gastrocnemius muscle and was thus labelled as EMG of the triceps surae muscle.

During the measurements the patient was seated in a semireclined chair. The chair and hydraulic system were individually adjusted for each patient so that the thigh was always horizontal, the knee flexed at 45° and ankle joint at 40° of plantar flexion. The range of ankle joint movements was 30° (15° of plantar, 15° of dorsal flexion).

First, the resistive torque during the plantar and dorsal flexion of the ankle joint was measured at four different frequencies of passive sinusoidal movements. Thereafter, the electrical stimulation was applied over the sural nerve for 20 min. Spasticity was measured immediately after the end of TENS (20 patients) and repeated 15 (20 patients), 30 (18 patients), 45 (15 patients) and 60 min (9 patients) following the end of TENS. In other words, the measurements were discontinued in those patients who found it hard to bear the protracted procedure and could not relax properly throughout the testing.

The possible contributions of the repeated passive movements of the foot to reduction of spasticity were excluded by performing the same experimental recordings without TENS, i.e. the 'placebo' stimulation (18).

The maximum value of resistive torque was measured as peak-to-peak amplitude regarding dorsal and plantar flexion of the foot at different frequencies (0.1, 0.5, 1 and 2 Hz) of passive ankle joint movements. Mean values and standard deviations of resistive torques were analysed statistically. Paired *t*-test was used for the comparison of the resistive torque values before and after TENS. The EMG activity was evaluated in relation to the phases of passive movements. The relative changes in EMG activity following the TENS application were also assessed.

RESULTS

In 18 of the 20 patients with hemiplegia or hemiparesis, statistically significant ($p < 0.005$) reduction of resistive torque was established immediately after the end of TENS at all frequencies of passive ankle joint movement (Fig. 1). The degree of this reduction varied between 2.7% and 39% in individual patients, with an average reduction of 12%. The variation of reduction was not related to the degree of resistive

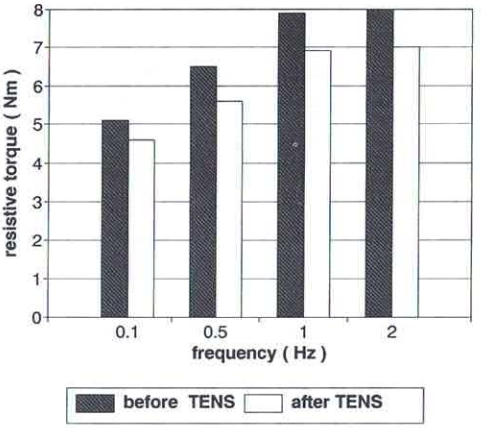

Fig. 1. Mean values of resistive torque at different frequencies of passive ankle joint movements (0.1, 0.5, 1 and 2 Hz) obtained before TENS and immediately after TENS in 20 patients with hemiplegia or hemiparesis.

torque in individual subjects before the stimulation was started.

As is evident from Fig. 1, the resistive torques were higher at higher frequencies of passive movement from 0.1 to 1 Hz, while no significant difference was observed between the values obtained at 1 and 2 Hz.

Fig. 2 presents the records of resistive torque, ankle joint angle and EMG activity during passive movement performed before and immediately following the end of 20 min TENS application.

The statistically significant reduction of resistive torque at all frequencies of passive ankle joint movements persisted 15 and 30 min ($p < 0.01$) as well as 45 min ($p < 0.05$) after TENS, whereas no statistically significant reduction could be discerned 60 min after TENS application.

The EMG activity was recorded in the triceps surae muscle during passive dorsal flexion and the activity of the tibialis anterior during passive plantar flexion of the foot (Fig. 2). The amplitude of the EMG activity of both muscle groups examined was increasing together with the increase in resistive torque at increasing frequencies of movement from 0.1 to 1 Hz, while no further increment was detected from 1 to 2 Hz. In 16 of 20 patients the activity of the triceps surae muscle was greater than that of the tibialis anterior muscle. EMG activity appeared even at slower movement frequencies, mostly in rather short bursts toward the end of the stretching period and was thus more of tonic than of phasic nature. A parallel decrease in EMG activity of one or both examined muscles and of the resistive torque following TENS application was observed in 15 of the 20 patients under investigation. In 5 patients, no significant changes in EMG activity were detected, while the resistive torque diminished after TENS.

DISCUSSION

The results of our study indicate that TENS, when

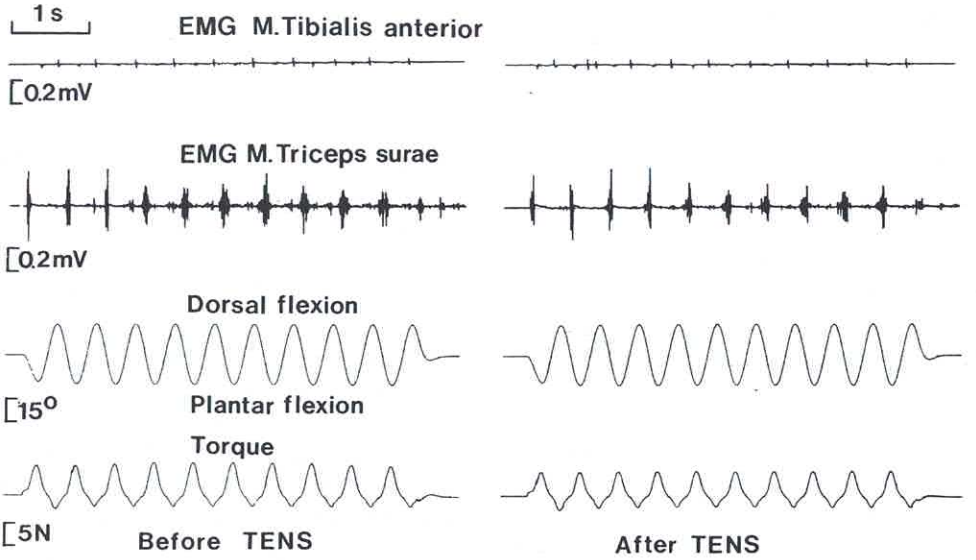

Fig. 2. Recording of EMG activity, ankle joint angle and resistive torque on sinusoidal passive movement (2 Hz) of the foot in a patient with spastic hemiplegia before (*left*) and immediately following TENS (*right*).

applied to the sural nerve area on the affected side in patients with chronic spastic hemiplegia, may cause a reduction of the exaggerated stretch reflex activity in spasticity. The decrease in resistive torques was statistically significant immediately following the end of TENS as well as after the measurements repeated up to 45 min later. The suppressive effect of a single TENS application on hemiplegic spasticity may last longer in some patients, though in our group of patients we were unable to discern any statistically significant prolonged effects which could have therapeutical implications.

The results of the present study are in agreement with the reports of Gregorič et al. (10) and Bajd et al. (3), who found that TENS applied to L2-L4 dermatomes reduced spasticity of the knee extensor muscles in some paraplegic patients, which is thus indicative of beneficial effects of TENS by reducing muscle hypertonia in both spinal and cerebral spasticity. TENS was effective to approximately the same as extent or even more so than electrical stimulation of the quadriceps muscles by functional electrical stimulation (FES), i.e. stimulation with higher intensity and lower frequency of pulses causing visible muscle contraction (3,25).

FES, which is related to muscle contraction, represents a combined afferent and efferent stimulation. The prolonged beneficial effects of FES on spasticity could be related at least partially to the stimulation of the cutaneous afferent nerve fibres. Conventional TENS, consisting of low-intensity and high-frequency stimulation, is believed to selectively activate large diameter sensory afferent fibres (13). TENS applied to the course of the sural nerve with an intensity just below the threshold of the minimal visible muscle contraction does not necessarily involve only the cutaneous nerve fibres; certain other large diameter nerve fibres might be excited too. However, the effects of a single application of TENS, not associated with muscle contraction, can be attributed only to the afferent effects of stimulation.

Spasticity is characterized by enhanced tonic stretch reflex, dependent on the velocity of muscle stretching, and by enhanced phasic stretch reflex (11), as was also confirmed in the present study. It was shown that different pathophysiological mechanisms, such as lack of presynaptic inhibition and abnormalities of interneurone activity, may be involved in spasticity (29). Segmental presynaptic inhibition can be enhanced by an increased afferent flow from the periphery (15). Thus, it could be claimed that in spastic patients with diminished suprasegmental presynaptic inhibition (5), the enhancement of weak physiological presynaptic mechanisms could be accomplished by reinforced segmental presynaptic inhibition (15). However, as shown in our study, the reduction of spasticity is not limited solely to the muscles innervated by the same spinal segment as stimulated by TENS. The transcutaneous stimulation on the sural nerve (S1 dermatome) reduced the spasticity in the triceps surae muscle (S1 myotome) as well as in tibialis anterior (L4/L5 myotome) (Fig. 2). The distant and bilateral effects of electrical stimulation on spasticity observed in some studies were attributed to 'centrifugal inhibition' (27) or to long–loop transcortical reflex mechanisms (2). These theories cannot be applied to the effects of stimulation observed in patients with complete spinal cord injuries below the level of the lesion (8). The beneficial therapeutic effects of afferent electrical stimulation include not only reduction of muscle hypertonia but also improvement of voluntary control (12, 16, 25). The mechanisms underlying a decrease in spasticity and improvement of motor functions following TENS application in hemiparetic subjects may be partly due to an enhancement of presynaptic inhibition of the spastic muscles and partly to a possible disinhibition of descending voluntary commands to the motoneurons of the paretic muscles (12). The observed widespread unspecific effects of electrical stimulation in the upper motor neuron syndromes might also be attributable to the plasticity of the central nervous system, which allows alterations of the excitation-inhibition balance (24).

The persistence of TENS effects on spasticity cannot be fully explained by the above-mentioned mechanisms. The effects of TENS observed in the present study persisted for up to 45 min after the end of stimulation. No significant effects were found 60 min after the end of TENS; however, the group of patients who completed the final test was probably too small to give statistically significant results. The long-lasting effects of TENS might be better explained by the production of endogenous substances which may follow peripheral electrical stimulation. An increased concentration of endorphines in CSF and blood was found in animals (29) and man (6, 23) after TENS of both high frequency and low intensity, implying that the opiate analgetic system might be activated by conventional TENS.

The results of the present study indicate that TENS can exert both immediate and short-term

post-stimulation suppressive effects on the exaggerated stretch reflex activity in cerebral spasticity, resulting in a mild to moderate decrease of muscle hypertonia. Further studies would be needed to explore the neurophysiological mechanisms underlying the long-lasting beneficial effects of afferent electrical stimulation on spasticity.

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Address for offprints:

Karmen Peterlin Potisk, MD
Institute for rehabilitation, Republic Slovenia
Linhartova 51
61000 Ljubljana
Slovenia